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identified as a key players in triggering a "resolution-to-inflammation" switch. Mature mast cells store a variety of cargoes (histamines, proteases, protease inhibitors) in their granules. Upon activation, these granule contents are mobilized, and can cause both local and systemic effects. Excessive or unbridled mast cell activation is a hallmark of systemic inflammatory diseases. There is substantial evidence that activation of mast cells in these diseases is essential to both the systemic inflammation and the specific organ injury. Recent studies have implicated mast cells in many organ-specific diseases, including chronic lung disease, arthritis, liver and kidney disease, urinary disease, atherosclerosis, and ocular diseases. Despite this recognized pathophysiological role of mast cells, no treatments for diseases that are associated with mast cell activation are currently available. The overall goal of this project is to establish the therapeutic role of mast cells in organ and tissue injury, and to unravel the molecular mechanism(s) by which mast cell activation mediates this response.

This study will examine the mechanisms of mast cell activation both in vitro and in vivo. Study in vitro will examine the roles of mast cells and granules in activating vascular endothelial cells, hepatocytes, podocytes, renal epithelial cells, lung cells, and lymphocytes. Study in vivo will test the hypothesis that mast cells are essential mediators of inflammation and tissue injury in models of asthma, myocarditis, glomerulonephritis, arthritis, and ocular disease. These studies will also seek to dissect the mechanism of mast cell activation. This analysis will address the roles of the mast cell protease-activated receptor and cytokines, of the classic and alternative activation pathways, and of endotoxin tolerance. These studies will provide fundamental information for the development of novel treatments for mast cell mediated diseases. PUBLIC